

9. Cohen PS, O'Brien TF, Schoenbaum SC, Medeiros AA: The risk of endothelial infection in adults with salmonella bacteremia. *Ann Intern Med* 1978; 89:931-932
10. Dupont JR, Bonavita JA, DiGiovanni RJ, Spector HB, Nelson SC: Acquired immunodeficiency syndrome and mycotic abdominal aortic aneurysms: A new challenge?—Report of a case. *J Vasc Surg* 1989; 10:254-257
11. Gouny P, Valverde A, Vincent D, et al: Human immunodeficiency virus and infected aneurysm of the abdominal aorta: Report of three cases. *Ann Vasc Surg* 1992; 6:239-243
12. Roussin-Bretagne S, Robert M, Tricot JF, et al: Abdominal aortic aneurysm caused by *Salmonella enteritidis* in a patient with human immunodeficiency virus (Letter). *Ann Med Interne (Paris)* 1991; 142:456-457
13. Mestres CA, Ninot S, de Lacy AM, et al: AIDS and *Salmonella*-infected abdominal aortic aneurysm. *Aust N Z J Surg* 1990; 60:225-226
14. Friedman SL: Gastrointestinal manifestations of the acquired immunodeficiency syndrome. In Sleisenger MH, Fordtran JS (Eds): *Gastrointestinal Disease: Pathophysiology, Diagnosis and Management*. Philadelphia, Pa, WB Saunders, 1993, pp 239-261
15. Centers for Disease Control: Update: Acquired immunodeficiency syndrome—United States. *MMWR* 1987; 36:522-526
16. Smith PD, Macher AM, Bookman MA, et al: *Salmonella typhimurium* enteritis and bacteremia in the acquired immunodeficiency syndrome. *Ann Intern Med* 1985; 102:207-209
17. Smith DF, Smith CC, Douglas JG, Reid TM, Gould IM: Severe salmonellosis related to oral administration of anti-diarrhoeal drugs. *Scott Med J* 1990; 35:176-177
18. Hoover DR, Saah AJ, Bacellar H, et al: Clinical manifestations of AIDS in the era of pneumocystis prophylaxis—Multicenter AIDS Cohort Study. *N Engl J Med* 1993; 329:1922-1926
19. Sabiston DC: Aortic abdominal aneurysms. In *Textbook of Surgery—The Biological Basis of Modern Surgical Practice*. Philadelphia, Pa, WB Saunders, 1991, pp 1566-1574
20. Fry WJ: Femoral artery aneurysms. In Sabiston DC (Ed): *Textbook of Surgery—The Biological Basis of Modern Surgical Practice*. Philadelphia, Pa, WB Saunders, 1991, pp 1574-1575
21. Reddy DJ, Smith RF, Elliot JP, Haddad GK, Wanek EA: Infected femoral artery aneurysms in drug addicts: Evolution of selective vascular reconstruction. *J Vasc Surg* 1986; 3:718-724
22. Johansen K, Devin J: Mycotic aortic aneurysms—A reappraisal. *Arch Surg* 1983; 118:583-588
23. Taylor LM Jr, Deitz DM, McConnell DB, Porter JM: Treatment of infected abdominal aneurysms by extraanatomic bypass, aneurysm excision, and drainage. *Am J Surg* 1988; 155:655-658

Prevention and Management of Hypernatremic Dehydration in Breast-fed Infants

LANCE A. CHILTON, MD
Albuquerque, New Mexico

SEVERAL REPORTS in the medical literature over the past 15 years have detailed the clinical course of critically ill breast-fed children with hypernatremic dehydration shortly after birth, all of whom required vigorous therapeutic efforts.¹⁻⁷ The child in the case reported here is such an infant, who became desperately ill with hypernatremic dehydration. Like some but not all of those in the cases reported, he seems to have recovered without sequelae.

(Chilton LA: Prevention and management of hypernatremic dehydration in breast-fed infants. *West J Med* 1995; 163:74-76)

From the Departments of Pediatrics, Lovelace Medical Center and the University of New Mexico School of Medicine, Albuquerque.

Reprint requests to Lance A. Chilton, MD, 2604 Candelaria Rd NW, Albuquerque, NM 87107.

Report of a Case

The patient was born to a 23-year-old gravida 1, para 0 woman at 41 weeks' gestation, weighing 2,843 grams after an uncomplicated pregnancy. Apgar scores were 8 at one minute and 9 at five minutes. The nursery stay was without complication, and the nurses' notes indicated that the mother was breast-feeding well. A circumcision was done when the infant was about 30 hours old, and mother and infant were discharged several hours later. Weight on the morning of discharge was 2,727 grams (decreased 4.8% from birth). As was usual for babies discharged within 48 hours, the child had an appointment scheduled three days later. At that visit, the mother expressed concern over the infant's fussiness during nursing and infrequent stools and urination. The findings of an examination were normal except for mild icterus (the serum bilirubin level was 227 μmol per liter [13.3 mg per dl]). The infant at this time weighed 2,560 grams (10% less than at birth). The mother strongly desired to breast-feed her infant and was given recommendations on infant care and feeding.

At 7 days of age, the patient was brought to the clinic by his parents who were concerned because he had had fever for about 8 hours and no urine output for approximately 24 hours. He was febrile, moderately lethargic, and markedly dehydrated, with dry mouth, dry sunken eyes, and a pronounced loss of skin turgor. He weighed 2,132 grams (24% below birth weight). The baby was moderately jaundiced.

Initial laboratory results included the following: serum sodium 182, potassium 3.5, chloride 138, and bicarbonate 25 mmol per liter; urea nitrogen (BUN), 73.2 mmol per liter (205 mg per dl); and creatinine, 212 μmol per liter (2.4 mg per dl). The leukocyte count was 9.0×10^9 per liter (9,000 per mm^3) with a normal differential; hemoglobin, 137 grams per liter (13.7 mg per dl); and platelet count, 321×10^9 per liter (321,000 per mm^3). A blood culture was later reported as negative for pathogens, as was the culture of the spinal fluid; the cerebrospinal fluid was xanthochromic (probably due to neonatal jaundice; the serum bilirubin level was 258 μmol per liter [15.1 mg per dl]), with 45 erythrocytes $\times 10^6$ per liter and 1 monocyte $\times 10^6$ per liter; the glucose level was 2.7 mmol per liter (48 mg per dl), and the protein level was 0.77 grams per liter (77 mg per dl).

An intravenous catheter was placed, and the child was given two boluses of 20 ml per kg of a solution of normal saline with 5% dextrose over the first hour. Antibiotics (ampicillin and gentamicin sulfate) were administered intravenously (they were discontinued two days later when the cultures were reported as negative). When the serum sodium level became available, the intravenous fluid was changed, first to dextrose with 0.675% sodium chloride and then to dextrose with 0.45% sodium chloride when no seizures occurred. The fluid infused was calculated to replace losses slowly over 24 hours to avoid rapid fluid shifts. Urine output

remained low during the first two days, but the child was intermittently active and alert. The serum sodium level fell slowly at first, presumably because of the relatively high sodium concentration of the infused fluid and the sodium accompanying the ampicillin. The serum sodium level fell to 178 mmol per liter 19 hours after admission, 171 mmol per liter on the second hospital day, 165 mmol per liter on the third, 158 mmol per liter on the fourth, and 154 mmol per liter on the fifth hospital day. Renal function study values fell more rapidly, with the BUN reaching 4.6 mmol per liter (13 mg per dl) and the creatinine 88.4 μ mol per liter (1.0 mg per dl) on the third hospital day. The fractional excretion of sodium on the second hospital day was 0.8, well within the range for prerenal azotemia. The urine osmolality on the second day was 472 mmol per kg (472 mOsm per kg), appearing to rule out diabetes insipidus. Specimens of the mother's breast milk were obtained about 36 hours after she had last breast-fed her infant; milk from the left breast had a measured sodium level of 78 mmol per liter, and from the right breast the value was 58 mmol per liter. Normal values for colostrum have been reported to be 22 ± 12 mmol per liter; for mature milk at two to three weeks, it is 7 ± 1 mmol per liter.⁸ The patient's mother appeared healthy and well hydrated and had no signs of malnutrition or cystic fibrosis, conditions that could elevate sodium levels. Her serum sodium level was not measured.

The infant's urine output increased by the end of the second hospital day, reaching 6 ml per kg per hour by the morning of the third day. After the breast milk sodium level became available, the mother decided not to breast-feed further. The medical staff, uncertain as to the likelihood of the problem recurring, did not attempt to convince her otherwise. Formula feeding was begun on the third hospital day, with the infant showing excellent weight gain before discharge.

The patient was discharged on the sixth hospital day with normal results on an examination and taking formula well. He has been seen for routine follow-up since, appearing to develop and grow normally. When last seen at 3 years of age, his height and weight were at the 35th and 50th percentiles, respectively. The patient's mother had decided against breast-feeding her second child, despite our assurance that a similar occurrence would be unlikely.

Discussion

Several issues arose in a sequential fashion in the treatment of this patient, starting with the urgent need to treat shock:

- How should immediate resuscitation be accomplished?
- What was the cause of the severe hypernatremia?
- What problems occurred in this breast-feeding dyad that led to the severe dehydration?
- What is the prognosis for this infant and others like him?

- Should the woman have resumed breast-feeding once her infant had been treated?

- What education should women new to breast-feeding receive before and after delivery?

- What fail-safe mechanisms should be in place to avoid possible disasters such as this?

It was clear on admission of this child that fluid resuscitation was urgent. Human plasma protein fraction (Plasmanate) or albumin might have been chosen; a normal saline solution is most readily available in such an emergency. We were fortunate to be able to establish an intravenous cannula almost immediately; otherwise, an intraosseous needle might have been placed. Because the presentation of a child in shock is not unlike that of septicemia, a workup for septicemia and initial treatment with antibiotics are often warranted.

When the infant's high serum sodium level was reported, the diagnosis of the cause of that condition became a priority, along with the careful observation of the infant and his serum sodium level to avoid the seizures that can occur from too rapid lowering of the sodium level.⁴ Several causes of hypernatremic dehydration have been listed⁹: some, such as cerebral injury, prolonged fever, salicylate toxicity, and hyperventilation, seemed unlikely with the history and the examination results. Diabetes insipidus was ruled out by the infant's ability to concentrate the urine to the degree expected for a newborn. Excessive salt intake remained the most likely possibility. Although cases of child abuse by salt poisoning have been reported, this seemed exceedingly unlikely with this family. Because there was no fluid intake other than breast milk, we measured the breast milk sodium concentration as the most likely source of a high sodium intake. The breast milk sodium level at 8 to 14 days averages 13.1 ± 0.6 (standard error of the mean) mmol per liter.⁸ The mother's breast milk sodium level at eight days measured 58 and 78 mmol per liter in the right and left breast, respectively. These measurements appeared to have established the cause of the hypernatremia; a fall in the sodium level has been documented each day after birth, making it likely that the level in this mother's milk was even higher on earlier days.⁸ One author studied 130 women who started breast-feeding, measuring breast milk sodium levels from day 3 to day 12.¹⁰ Women who failed at breast-feeding did not have the same drop in breast milk sodium values seen in women who succeeded. Thus, in the case reported here, the mother's high breast milk sodium values may have indicated that she would not have succeeded at breast-feeding.

How did the severe dehydration develop? We do not have a clear answer to this question. Motivation was not a problem: events before, during, and long after the crisis have convinced us that the patient's mother cared deeply for him and was strongly motivated to breast-feed him successfully. By history, she was feeding him frequently enough (every 2 to 3 hours). She apparently was eating and drinking sufficiently. How well did the family know the signs of dehydration and severe illness?

Apparently not well enough. Should we have recognized the impending problem at the first clinic visit at 3 days of age? According to some authors, a 10% weight loss is well within 2 standard deviations of the mean maximal weight loss ($5.8\% \pm 3.2\%$) for a breast-feeding infant.¹¹

The prognosis for infants with severe dehydration has generally been good, but not invariably so. A case has been reported of an infant with a serum sodium level of 189 mmol per liter who had a normal Bayley Infant Mental and Motor Development Index at age 11 months.⁶ Other authors, in reporting on four breast-fed infants with severe dehydration (3 of whom had serum sodium levels ranging from 173 to 190 mmol per liter), found all to have normal development at 2 years, though one had nystagmus.⁵ On the other hand, the course of an infant was described who presented with severe dehydration and a serum sodium level of 176 mmol per liter; that child has had persistent seizures and severe developmental delay, although the child's premorbid condition was not known.⁷ A good neurologic outcome has been reported in another infant, although elevated blood pressures persisted in that patient through the period of follow-up.³ Our patient appears to have done well.

One of the most difficult questions in this case is whether the mother should resume breast-feeding. The issue of being able to breast-feed once a high sodium level has been detected in the milk has not been addressed.¹⁰ Will the milk continue to be high in sodium, placing the infant again at a high risk of hypernatremia? In the case of hypernatremic dehydration in a breast-fed infant,⁷ maternal milk sodium levels were twice normal (16 mmol per liter) at 30 days; the author recommended against allowing the child to return to the breast. Another author, however, noted a rapid fall in breast milk sodium to normal values by about 21 days, and stated the following^{6(p372)}:

Because the primary cause of the disorder [hypernatremic dehydration] is probably insufficient lactation and not elevated sodium in the breast milk, therapy should include supporting the mother and giving her the opportunity to successfully relactate. . . . Relactation can be accomplished physiologically through frequent breast pumping and supplementation using a nursing supplementer while the infant provides sucking stimulation to the breasts.

Unfortunately, in this situation, we were uncertain of the danger to the infant if the mother resumed breast-feeding; by the time we found Thullen's paper, the mother, sensing our indecision, had decided that she no longer wished to breast-feed her infant.

What education should women new to breast-feeding receive before and after delivery? Our nursery and labor and delivery nursing staffs are uniformly supportive of women breast-feeding their infants, as is the prepartum maternal education staff. Women are urged to breast-feed, and a high percentage (about 75%) of women leave the hospital nursing their infants. They are given the telephone numbers of hospital staff members especially knowledgeable about breast-feeding to call should problems arise. Hospital stays here, however, as elsewhere, have continued to decrease after delivery, now averaging

less than 24 hours for women after a vaginal delivery and less than 3 days for a woman who has had a cesarean delivery. This allows little time for education or for ensuring that breast-feeding is well established.

Because we have seen several cases such as this one, we have instituted a policy of telephoning all new mothers one to two days after discharge to attempt to detect situations where nursing problems are occurring. Our hospital has hired a certified lactation consultant to provide education and support to breast-feeding women and to head off impending problems such as this one.

The following guidelines have been published for detecting possibly hypernatremic dehydrated infants⁷: slow feeding, poor sucking; a sleepy, quiet, "good baby," causing parents to be unaware of evolving dehydration; and possibly diminished milk secretion. Our first face-to-face visit with new mothers is five to ten days after discharge; the presence of these findings or the irritability that others note in hypernatremic infants prompts the practitioner to ask the parent to bring the infant to the clinic earlier than this first scheduled visit.

How can we prevent possible disasters like this? Lacking the ability to keep women and their infants in the hospital long enough to observe breast-feeding adequately and to educate them fully regarding the signs of dehydration and hypernatremia, steps such as those referred to earlier are of considerable importance. More prenatal education, written information as to warning signs, follow-up calls, early postpartum visits, and round-the-clock availability of infant medical care may help. All of us—physicians, nurses, lactation consultants, and others—need to continue to make the case to all mothers that "breast is best," despite such rare, untoward events as this.¹²

Acknowledgment

Annie Furie, RN, IBCLC, and D. Wacondo, LPN, provided advice on breast-feeding. Larry Berger, MD, made many constructive suggestions in reviewing this article in manuscript form.

REFERENCES

1. Jaffe KM, Kraemer MJ, Robison MC: Hypernatremia in breast-fed newborns. *West J Med* 1981; 135:54-55
2. Rowland TW, Zori RT, Lafleur WR: Malnutrition and hypernatremic dehydration in breast-fed infants. *JAMA* 1982; 247:1016-1017
3. Clarke TA, Markarian M, Griswold W, et al: Hypernatremic dehydration resulting from inadequate breast-feeding. *Pediatrics* 1979; 63:931-932
4. Marino R, Gourji S, Rosenfeld W: Neonatal metabolic casebook: Hypernatremia and breast feeding. *J Perinatol* 1989; 9:451-453
5. Roddey ES Jr, Swetenburg RL: Critical weight loss and malnutrition in breast-fed infants. *Am J Dis Child* 1981; 135:597-599
6. Thullen JD: Management of hypernatremic dehydration due to insufficient lactation. *Clin Pediatr* 1988; 27:370-372
7. Peters JM: Hypernatremia in breast-fed infants due to elevated breast milk sodium. *J Am Osteopath Assoc* 1989; 89:1165-1170
8. Koo WWK, Gupta JM: Breast milk sodium. *Arch Dis Child* 1982; 57:500-502
9. Finberg L: Hypernatremic dehydration. *Adv Pediatr* 1969; 16:325-341
10. Morton JA: The clinical usefulness of breast milk sodium in the assessment of lactogenesis. *Pediatrics* 1994; 93:802-806
11. Maisels MJ, Gifford K: Breast-feeding, weight loss, and jaundice. *J Pediatr* 1983; 102:117-118
12. Berger L: When should one discourage breast-feeding? *Pediatrics* 1981; 67:300-301